*DISCUSSION**

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THIS symposium has been most stimulating. To tie in more closely with its content, I shall shorten such discussion as I had prepared and enlarge it in another direction.

The striking aspect of the results of surgical treatment of epilepsy is that, as with many other surgical therapies, success in the eradication of intractable focal epilepsy is a function of the selection of patients. This is reflected in the series presented this evening by Dr. Ransohoff and Dr. Pool, in which, in agreement with Penfield[1] and Falconer[2] and their associates, and others, cessation of seizures was achieved most frequently in patients harboring demonstrable morphologic lesions of the excised temporal lobe tissue.

Both the Montreal and the Maudsley groups have successfully devoted much of their effort to the development of criteria for the preoperative selection of such patients, that is, of criteria which would not only identify the cases which can be operated upon, but which would also specify as closely as possible the postoperative prognosis for the elimination of seizures. Since these criteria are well known to this audience, I shall say no more about them and direct my comments to a criterion which has received little or no explicit consideration, namely, the timing of neurosurgical intervention in the operable case of intractable focal epilepsy.

I have long admired the tradition of giving the conclusions first and I should like to follow it now. I would like to show that in cases with intractable focal epilepsy, there may come a time when it is too late for neurosurgical treatment.

Data from our recent experience at the Albert Einstein College of Medicine have indicated the possibility that criteria based on observation of the electrographic evolution of focal epilepsy may be used in conjunction with the response of the patient's seizures to anticonvulsant measures. In the past three years, 16 patients with intractable seizures

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due to atrophic lesions of the brain, have been studied intensively with routine EEGs, basal leads, activation procedures and intracarotid amobarbital injections, in collaboration with Dr. Robert Katzman and Miss Molly Cox. Eight patients did come to operation subsequently.

In 15 of the 16 patients it was possible to secure documented electrographic perspective spanning a period of up to 12 years, and not less than 18 months. The EEG tracings of these patients were drawn from the files of the EEG laboratory of the Bronx Municipal Hospital Center, or were borrowed for review from other institutions previously caring for these patients.

Longitudinal review of the electrographic evidence in each patient revealed that in six of the 13 adults, and in the two patients under 14 years of age, there was clear evidence of progression of focal abnormality to involve homotopic areas of the contralateral hemisphere. In five other patients over 14 years of age, the electrographic abnormality was static. In three of these, there was bilateral abnormality involving the temporal areas, with evidence suggesting one-sided preponderance. With evidence of progression in hand from the previous group of cases, it is difficult to resist the speculation that these not-quite-symmetric bitemporal cases may have been unitemporal at an earlier stage of their illness.

Thus, among the 16 patients with intractable seizures in whom a detailed electrographic work-up was carried out to determine operability, there was evidence of EEG progression in eight.

Our material does not contain any information as to the probability of the development of what may be called "progressive cortical epilepsy" among patients with presently unifocal epilepsy. This question is now being studied.

In this symposium, Dr. Goldensohn and Dr. Purpura have reminded us of the progressive aspect of focal epilepsy with experimental records from the cat. Awareness of the capacity of focal epileptogenic lesions to produce "mirror foci" has existed since the introduction of methods for the creation of chronic experimental epileptogenic lesions 20 years ago.[3] Nevertheless, in the clinical perspective up to now, focal cortical epilepsy continues to be regarded predominantly as a circumscribed process, originating from one or more usually static, or occasionally migratory loci. I think that we have all had the experience of following patients with unifocal epilepsy for long periods of time without observing any extension to contralateral homotopic areas. Absence of progres-

sion has been noted recently by Bloom, Jasper and Rasmussen.[4] There is also probably a species difference between epileptic man, who seems to form projected foci late, and apparently infrequently, and the epileptic rabbits and cats of Morrell[5] and the epileptic monkeys of Kopeloff,[3] and of Pope[6] and their associates, in which mirror foci develop early and lastingly.

The stage of development of the progressive EEG picture influenced the determination of operability in our patients. Thus, beginning involvement of the contralateral temporal lobe led to surgical intervention in one case, with successful postoperative outcome. Other patients, in whom a lateralized preponderance could not be demonstrated, were regarded as unlikely to benefit from operation.[4] Efforts to control seizures with anti-convulsants were maintained.

The findings in these sixteen cases emphasize the importance of following the intractable focal epileptic cases with electroencephalographic studies at regular intervals. These findings also add the dimension of time to the criteria of selection of patients for optimum benefit from neurosurgical treatment.

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